Abstract

Apart from clinical, histological and biochemical indices, genomics are now being employed to unravel the pathogenetic mechanisms in the disease progression of IgA nephritis (IgAN). The results of angiotensin converting enzyme (ACE) gene polymorphism have been controversial. Those patients with the DD genotype seem to have a poorer prognosis. However, with high dose angiotensin receptor blocker (ARB) therapy, the ACE gene polymorphism status of a patient may no longer be a matter for concern as those with the DD genotype would also respond favourably to high dose ARB therapy. Association studies with gene sequencing and haplotypes have suggested that multiple genes are involved in the pathogenesis of IgAN. Some workers have reported a synergistic effect in the combined analysis of AGT-M235T and ACE I/D polymorphism. With the use of deoxyribonucleic acid (DNA) microarray, tens of thousands of gene expressions genome-wide can be examined together simultaneously. A locus of familial IgAN has been described with strong evidence of linkage to IgAN1 on chromosome 6q22-23. Two other loci were reported at 4q26-31 and 17q12-22. DNA microarray techniques could also help in the identification of specific pathogenic genes that are up- or down-regulated and this may allow genome wide analyses of these genes and their role in the pathogenesis and progression of IgAN. Recently, using genome-wide association studies (GWAS) more loci for disease susceptibility for IgAN have been identified at 17p13, 8p23, 22q12, 1q32 and 6p21.

Key words: Gene sequencing, Haplotypes, Microarray, Single nucleotide polymorphism